Vascular damage mediates neuronal and non-neuronal pathology following short and long-term rotenone administration in Sprague-Dawley rats

Khaled Radad, Khaled Hassanein, Rudolf Moldzio, Wolf Dieter Rausch

Abstract:

Even though rotenone has been used extensively in recent years to produce a model of Parkinson disease in rats, its systemic effects either on neurons apart from dopaminergic structures or non-neuronal tissues have not been elucidated well. In our present study, 30 adult Sprague-Dawley rats were divided into three even groups. A short-term rotenone-treated group received 10mg/kg b.w. rotenone daily for 7 days. The long-term rotenone-treated group received 3mg/kg b.w. rotenone daily for 30 days. The control group received vehicle only and were kept 5 rats each in parallel to both short- and long-term rotenone treated groups. It was found that short-term rotenone treatment produced marked vascular damages associated with ischemic neuronal degeneration particularly in the thalamus, cerebellum and nucleus dentatus. In long-term rotenone-treated group, vascular changes were less severe and neuronal degeneration was associated with mild microglial proliferation and astrocytosis. Non-neuronal pathology as the result of short-term rotenone exposure consisted of degeneration and necrosis of seminiferous tubular epithelia with formation of spermatide multinucleate giant cells. On the other hand, long-term rotenone treatment did not affect testicles and only caused sinusoidal dilatation in the liver, myocardial degeneration in the heart and interstitial hemorrhages in the kidneys and lungs. In conclusions, damage to blood vasculature by rotenone appeared mediating neuronal and non-neuronal pathology in Sprague-Dawley rats. This effect might provide new insights for ethiopathogenesis of neurodegenerative diseases and contributes to the understanding of hemorrhagic stroke.Even though rotenone has been used extensively in recent years to produce a model of Parkinson disease in rats, its systemic effects either on neurons apart from dopaminergic structures or non-neuronal tissues have not been elucidated well. In our present study, 30 adult Sprague-Dawley rats were divided into three even groups. A short-term rotenone-treated group received 10mg/kg b.w. rotenone daily for 7 days. The long-term rotenone-treated group received 3mg/kg b.w. rotenone daily for 30 days. The control group received vehicle only and were kept 5 rats each in parallel to both short- and long-term rotenone treated groups. It was found that short-term rotenone treatment produced marked vascular damages associated with ischemic neuronal degeneration particularly in the thalamus, cerebellum and nucleus dentatus. In long-term rotenone-treated group, vascular changes were less severe and neuronal degeneration was associated with mild microglial proliferation and astrocytosis. Non-neuronal pathology as the result of short-term rotenone exposure consisted of degeneration and necrosis of seminiferous tubular epithelia with formation of spermatide multinucleate giant cells. On the other hand, long-term rotenone treatment did not affect testicles and only caused sinusoidal dilatation in the liver, myocardial degeneration in the heart and interstitial hemorrhages in the kidneys and lungs. In conclusions, damage to blood vasculature by rotenone appeared mediating neuronal and non-neuronal pathology in Sprague-Dawley rats. This effect might provide new insights for ethiopathogenesis of neurodegenerative diseases and contributes to the understanding of hemorrhagic stroke.

Keywords:

Brain; Degeneration; Histopathology; Rats; Rotenone; Vascular

Published In:

Experimental and Toxicologic Pathology , 65 (1) , 41-47